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15 **UNITED STATES DISTRICT COURT**  
16 **FOR THE NORTHERN DISTRICT OF CALIFORNIA**  
17 **SAN FRANCISCO DIVISION**

18 IN RE: BABY FOOD PRODUCTS LIABILITY  
LITIGATION

Case No. 24-md-3101-JSC  
MDL 3101

19  
20 This Document Relates to:  
21  
22 ALL ACTIONS

Hon. Jacqueline Scott Corley

23 **REPLY IN SUPPORT OF**  
**DEFENDANTS' MOTION TO EXCLUDE**  
**PLAINTIFFS' CAUSATION/**  
**EPIDEMIOLOGY EXPERTS**

24 Date: December 8, 2025

25 Time: 9:00 a.m.

26 Location: Courtroom 8

19th Floor 450 Golden Gate Ave.  
San Francisco, CA 94102

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1 **I. INTRODUCTION**

2 Plaintiffs' Opposition confirms that their experts do not—and cannot—offer reliable  
 3 scientific evidence to answer the general causation question: can eating Defendants' baby food  
 4 products cause autism or ADHD? Their experts' opinions rest on multiple methodologic errors and  
 5 analytical gaps. They start with a lawyer-generated exposure model that the experts admit does not  
 6 reflect realistic consumption of baby foods; they then rely on studies of lead and arsenic exposure  
 7 that do not involve food at all; and finally, they attempt to extrapolate from those studies even though  
 8 they are not designed to answer and cannot answer *any* (let alone the correct) cause and effect  
 9 question. Each of these methodological flaws renders their general causation opinions unreliable.

10 Plaintiffs continue to dodge the central methodological flaw in these experts' opinions: **zero**  
 11 scientific studies assess whether eating baby food (or any food) can cause autism or ADHD. *See*  
 12 Defs.' Br. No. 3, at 7-8.<sup>1</sup> Instead, Plaintiffs move the goalposts, calling lead and arsenic the "toxic  
 13 agents at issue." Opp. at 64. But the products at issue in this litigation are not lead and arsenic; they  
 14 are hundreds of baby food products made of fruits, vegetables, and grains that are widely recognized  
 15 as essential to healthy neurodevelopment. *See* Defs.' Br. No. 3, at 9-10. Plaintiffs' repeated attempts  
 16 to treat baby foods like radiation, asbestos, or paint chips lack any basis in science. *See* Opp. at 66.  
 17 Unlike those substances, baby foods are complex mixtures rich in nutrients and other constituents  
 18 that impede the absorption and retention of lead and arsenic, as well as the potential toxicity of any  
 19 trace metals contained in the food. And food like Defendants' baby food is known to be **good** (in  
 20 fact, essential) for children's developing brains. Because of these undisputed scientific facts, the  
 21 absence of any science tied to food and showing that eating these types of foods has a negative  
 22 effect—specifically by causing autism and ADHD—is a gap that Plaintiffs experts fail to bridge.

23 With no data supporting their opinion with respect to the products at issue—food—Plaintiffs  
 24 are left to argue that their experts nevertheless survive Rule 702 because their opinions depend on  
 25

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26 <sup>1</sup> "Defs.' Br. No. 1" refers to Defendants' Background Brief in Support of their Motion to Exclude  
 27 Plaintiffs' General Causation Experts (Brief 1), Dkt. No. 611; "Defs.' Br. No. 2" refers to  
 28 Defendants' Brief in Support of Joint Motion to Exclude Plaintiffs' Exposure Experts Rachel Jones  
 and Priscilla Barr, Dkt. No. 612; "Defs.' Br. No. 3" refers to Defendants' Motion to Exclude  
 Plaintiffs' Causation/Epidemiology Experts (Brief 3), Dkt. No. 614.

1 non-food studies that, according to Plaintiffs, show causal associations with lead and arsenic  
 2 exposure at supposedly food-relevant doses. But before Plaintiffs' experts can extrapolate from  
 3 literature that does not involve baby food (or any food) to opine that all of Defendants' baby food  
 4 products can cause autism or ADHD, Rule 702 requires that they demonstrate the reliability of their  
 5 extrapolation method. They fail to do so. None of Plaintiffs' causation experts give more than a  
 6 perfunctory nod to the lawyer-created exposure analyses Dr. Jones and Ms. Barr sponsor. Indeed,  
 7 some of Plaintiffs' experts did not even look at the analyses and others used them merely as a  
 8 springboard for an opinion that "any dose" of lead and arsenic can cause these conditions.

9       Additionally, Plaintiffs fail to adequately connect Dr. Jones' calculations to relevant studies;  
 10 they point to only four studies that they say involve "doses" matching Dr. Jones' calculations. But  
 11 one study is not even in humans at any dose—it is in zebrafish swimming in pools of arsenic-infused  
 12 water. The others measure biomarkers of exposure, not dose, and are rife with methodological gaps  
 13 ranging from not evaluating children who are the relevant age, to not providing data on how dose  
 14 relates (if at all) to increased risk, to not reporting any statistically significant results.

15       In the end, Plaintiffs try to turn science into a study-counting exercise. They argue that  
 16 "hundreds" of papers report "associations" between lead and arsenic and "symptoms that would be  
 17 diagnosed as ASD or ADHD." Opp. at 9, 24. In doing so, they not only improperly extrapolate from  
 18 studies that do not investigate the injuries alleged in this MDL (diagnosed autism and ADHD), they  
 19 also rely on exposure windows and populations not relevant to this case. The vast majority of the  
 20 papers on which they rely measure exposures *after* the diagnosis of the disorder or the manifestation  
 21 of the symptoms being studied. The Reference Manual and all other authorities make clear that such  
 22 studies cannot reliably support a causation opinion. And the studies lacking this requirement for  
 23 "causal contribution" are not some tiny fraction of the literature on which Plaintiffs' experts rely:  
 24 ***nearly 90% of these studies cannot satisfy the basic requirement of temporality.***

25       Finally, in a sleight of hand, Plaintiffs suggest that it is the "consensus" of the scientific  
 26 community that autism is caused by both genetic and "environmental" factors. *See* Opp. at 6-8. But  
 27 for autism researchers, "environmental" factors include anything that is non-genetic, such as  
 28 advanced parental age, one of the few known non-genetic risk factors for autism. *See* Defs.' Br. No.

1, at 8. Notably, ***none*** of the publications Plaintiffs cite suggest that exposure to the trace amounts  
 2 of lead and arsenic in baby food products can cause autism or ADHD. Rather, all medical  
 3 organizations and public health authorities advise parents to feed their children fruits, vegetables,  
 4 and grains of the type found in commercial baby foods—precisely because the vitamins and  
 5 nutrients they contain are essential for healthy brain development.<sup>2</sup> *Id.* at 9-10.

6 The question here is not, as Plaintiffs suggest, “what is good peer-reviewed science (and  
 7 what is not).” Opp. at 3. The question is whether the peer-reviewed science Plaintiffs’ experts cite  
 8 can reliably answer the general causation question in this case, and reliably support an opinion that  
 9 eating apples, blueberries, carrots, spinach, and rice—foods broadly recognized as essential for  
 10 healthy brain development—can cause autism or ADHD. The answer to ***that*** question is no.  
 11 Plaintiffs’ causation experts’ opinions should, therefore, be excluded under Rule 702.<sup>3</sup>

## 12 **II. ARGUMENT**

### 13 **A. Plaintiffs’ Experts’ Opinions Suffer From A Fundamental Flaw: They Are Not 14 Based on Food Studies.**

15 Plaintiffs do not directly respond to Defendants’ arguments that: (1) Plaintiffs’ experts’ baby  
 16 food opinions are not generally accepted in the medical and scientific community; (2) ***no*** studies  
 17 link baby food to an increased risk of autism or ADHD; (3) Drs. Ritz, Hu, Aschner, and Shapiro  
 18 agree that, because food is a complex mixture, if a scientist wants to know what effects particular  
 19 foods have on human health she must study those foods; (4) the methodology employed by the  
 20 experts here is different from what they use in their academic work, where they study food to reach  
 21 conclusions about food; and (5) none of Plaintiffs’ experts considered, let alone demonstrated, that  
 22 the net effect of all the nutrients and other elements in baby food is to cause autism and ADHD at a  
 23 population level. These analytical gaps and methodological flaws, to which Plaintiffs offer no direct

---

24  
 25 <sup>2</sup> Plaintiffs ignore entirely that the American Academy of Pediatrics has just this year made clear  
 26 that autism “is complex, highly variable and ***increasingly linked to genetics.***” American Academy  
 27 of Pediatrics, AAP Statement on White House Autism Announcement (Sept. 22, 2025),  
<https://www.aap.org/en/news-room/news-releases/aap/2025/aap-statement-on-white-house-autism-announcement>.

28 <sup>3</sup> Footnote 2 of Plaintiffs’ Omnibus Opposition to Defendants’ Motions to Exclude is incorrect in  
 that it cites activity by a manufacturer that is not a party to this MDL and is a different entity from  
 the Defendant in this case Sprout Foods, Inc.

1 response, undermine the reliability of their experts' opinions and compel exclusion.

2 The arguments Plaintiffs *do* make rely on their experts' *ipse dixit* and circular reasoning.

3 Plaintiffs' experts do not make any effort to prove harm from food because they do not believe they  
 4 need to prove harm from food. Opp. at 64 (calling lack of food studies a "red herring" because the  
 5 "toxic agent" is "lead and arsenic," not food); *see also* Kiser Decl., Ex. 46, at 145:19-22 (Gardener  
 6 Landon Tr.) (explaining that because her "report has to do with heavy metals," Dr. Gardener treated  
 7 "foods independent of heavy metals" as "beyond the scope."); Defs.' Br. No. 3, at 10-15.<sup>4</sup> But  
 8 Plaintiffs' experts fail to test their beliefs with relevant scientific data. In fact, Plaintiffs' opposition  
 9 confirms that their experts deliberately ignored the most obvious question here: Given that lead and  
 10 arsenic are known to be naturally present to some degree in most fruits, vegetables, and grains as a  
 11 function of the growing process, and given the known benefits of fruits, vegetables, and grains for  
 12 healthy development, including brain development, is there reliable population-level evidence that  
 13 these foods actually harm neurodevelopment by causing autism and/or ADHD? Defs.' Br. No. 3, at  
 14 9-10, 13-15. Plaintiffs' experts' choice to ignore evidence universally accepted by scientists  
 15 regarding the very nature and impact of healthy food (despite its potential to have trace levels of  
 16 lead or arsenic) is the essence of an unsound and result-driven methodology under Rule 702. *In re*  
 17 *Lipitor (Atorvastatin Calcium) Mtg., Sales Pracs. & Prod. Liab. Litig. (No. II) MDL 2502*, 892 F.3d  
 18 624, 634 (4th Cir. 2018); *see also* Defs' Br. No. 3, at 9-10, 16-18.

19 **1. Plaintiffs' Argument That Food Studies Are Unnecessary Contradicts**  
**20 Both the Literature They Cite and Their Own Experts.**

21 Plaintiffs offer two arguments to justify treating "lead and arsenic,"—not Defendants' baby  
 22 foods—as the "toxic agent" at issue. First, they incorrectly claim that there are studies causally  
 23

24 \_\_\_\_\_  
 25 <sup>4</sup> Documents cited as "Pltfs. Ex." refer to the exhibits filed at Dkt. No. 642-1, which accompany the  
 26 Declaration of Aimee H. Wagstaff in Support of Plaintiffs' Omnibus Opposition to Defendants'  
 27 Motions to Exclude. Documents cited as "Kiser Decl., Ex." refer to the exhibits filed at Dkt. No.  
 28 611-1, which accompany the Declaration of Livia M. Kiser in Support of Defendants' Motion to  
 Exclude Plaintiffs' General Causation Experts. All other exhibit citations refer to the documents  
 submitted with the contemporaneously filed Declaration of Brooke Killian Kim in Support of  
 Defendants' Reply in Support of Defendants' Motion to Exclude Plaintiffs' Causation/Epidemiology Experts ("Kim Decl. Ex.").

linking lead and arsenic to autism, and lead to ADHD, and that those studies necessarily include food.<sup>5</sup> Second, they assert that route of exposure to lead and arsenic is “irrelevant” because “[t]he primary systemic toxic effects of [lead] are the same regardless of the route of entry into the body.” Opp. at 66 (citing Pltfs. Ex. 26, at 12 (ATSDR Lead Profile)). But the authorities on which Plaintiffs rely do not support their position, and their own experts disavow these arguments.

Plaintiffs cite Nehzomi (2024) as proving the alleged acceptance of a causal link between autism and lead and arsenic, and the application of that conclusion to healthy food—including Defendants’ baby food. But Nehzomi is a review article that covers various potential environmental contaminants beyond heavy metals, provides no original scientific research, and does not examine any studies linking autism to food (because there are none). *See* Pltfs. Ex. 81 (Nehzomi (2024)). Plus, Plaintiffs omit from their discussion of Nehzomi that the authors expressly state that there are “discrepancies among research findings,” and that “causation remains debated.” *Id.* at 7. The authors also suggest that further research should particularly focus on the prenatal window—not the postnatal period. *Id.*<sup>6</sup> That Plaintiffs offer only Nehzomi to justify their experts’ methods for

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<sup>5</sup> Defendants’ full discussion of the general epidemiologic literature on which Plaintiffs rely is in Section II(D), below. Remarkable and worth noting here, however, is Plaintiffs’ argument that the general epidemiologic studies assess food exposure, because the children had to have been eating food and that food had to have contributed to their levels of heavy metals. That argument only underscores how alarmingly unscientific and results-oriented Plaintiffs’ experts’ opinions are. All children in the world eat food—including children without symptoms of autism or ADHD in the studies Plaintiffs’ experts cite. But none of the studies control for different dietary habits between affected and unaffected children or investigate food as a source of exposure. Thus, no expert can reliably argue that this general literature speaks to whether lead or arsenic in food increases the risk of autism or ADHD. Indeed, when Dr. Ritz first testified in this litigation in 2021, and cited the same epidemiologic studies, she agreed that studies about whether food consumption causes autism or ADHD “don’t exist.” Pltfs. Ex. 24, at 12:13-13:25 (Ritz N.C. Tr. Vol. I).

<sup>6</sup> A full quote of the relevant paragraph is set forth below:

Epidemiological studies have found correlations between elevated lead levels and ASD, though ***causation remains debated***. The timing of exposure, especially prenatal, is crucial in determining its impact on neurodevelopment. Further research is needed to fully understand these relationships. Studies on the impact of lead exposure on ASD development have ***yielded inconsistent and conflicting results***. Some research suggests that prenatal exposure to lead could be a contributing factor to ASD onset in children and that children with ASD have notably higher levels of

1 reaching causation opinions betrays the key gap in their experts' analysis. Surely, if Plaintiffs had  
 2 actual studies showing that food can cause autism and ADHD as a result of trace levels of any heavy  
 3 metals, they would offer it. They do not because they cannot. Plaintiffs are improperly asking this  
 4 Court to have the law lead science, not follow it. *See Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319  
 5 (7th Cir. 1996).

6 Similarly unsupported is Plaintiffs' argument that "dietary heavy metal exposure poses a  
 7 higher health risk than other sources" because food is a driver of BLLs in children 1-6 years old.  
 8 Opp. at 64 (citing Kiser Decl., Ex. 7, at 85 (Gardener Rep.) (citing Pltfs. Ex. 80 (Zartarian (2017))).  
 9 First, Plaintiffs cite no authority supporting their speculation that food "poses a higher health risk"—  
 10 whatever that vague *ipse dixit* means. Second, Zartarian (2017) is not a food study and does not  
 11 claim that food meaningfully contributes to elevated BLLs in U.S. children below age two. Pltfs.  
 12 Ex. 80, at 4 (Zartarian (2017)). Rather, the paper's focus is on modeling lead exposure from water  
 13 and finds: "For 0- to-6-mo-olds, soil/dust and water ingestion pathways predominate at the highest  
 14 BLL percentiles" and for "1- to <2 y-olds, soil/dust ingestion was the dominant pathway above the  
 15 ~80th BLL percentile." *Id.* at 4. Also, "the pathway contributions for 2- to <6-y-olds were essentially  
 16 the same as for 1- to-<2-y-olds." *Id.*; *see also* Opp. at 64.

17 Plaintiffs ignore the testimony of their own toxicologist, Dr. Aschner, who agreed that the  
 18 general proposition set out in the ATSDR, that the primary effects of lead can occur through any  
 19 route of exposure, cannot be stretched to mean that there is no difference between eating paint chips  
 20 and eating carrots. *See contra* Opp. at 64, 66. Dr. Aschner agreed that "there are differences of  
 21 absorption between lead and the other metals" where route of exposure "is in liquid media, in water,  
 22 for example, versus food." Kiser Decl., Ex. 47, at 24:25-25:20 (Aschner Landon Tr.).

23  
 24 urinary and hair lead compared to control subjects. However, ***other studies indicate***  
 25 ***that increased lead levels do not necessarily correlate with a heightened presence***  
 26 ***of the core ASD symptoms*** outlined in the Diagnostic and Statistical Manual of  
 27 Mental Disorders (DSM-IV) criteria. The ***discrepancies among research findings***  
 28 ***regarding lead exposure could be attributed to variations in geographical exposure***  
***to lead or potentially to socio-demographic factors or clinical characteristics*** within  
 the study population.

Pltfs. Ex. 81, at 7 (Nehzomi (2024)) (emphases added) (internal citations omitted).

1       Other Plaintiffs' experts agreed that exposure to lead or arsenic through food is different  
 2 from other sources, given the beneficial constituents in food. Dr. Shapiro, for example, explained  
 3 that "heavy metal absorption may be different based on the presence or absence of certain nutrients"  
 4 and so when considering "how food contributed to someone's neurodevelopment, you do have to  
 5 consider all the components of that food." Kiser Decl., Ex. 48, at 353:1-10, 353:25-354:14 (Shapiro  
 6 Landon Tr., Vol. I). Similarly, Dr. Ritz has previously conceded that total food intake may influence  
 7 susceptibility to lead's neurological effects. Kiser Decl., Ex. 43 at 88:15-89:1 (Ritz Landon Tr.).  
 8 She also testified that she "could imagine" that the bioavailability of lead in food (how much can be  
 9 released from the food matrix and thus be absorbable via the gut) "has to do with the consistency of  
 10 the product" consumed—but she admitted that was not her area of expertise. *Id.* at 71:15-23.

11       Plaintiffs also cannot rest on Dr. Hu's opinion that "[o]nce lead is absorbed from baby food,  
 12 it would not be expected to behave or carry risks of causing adverse neurodevelopmental outcomes,  
 13 including ASD, any differently from lead that is absorbed from any other source." *See Opp.* at 64  
 14 (quoting Kiser Decl., Ex. 4, at 35 (Hu Rep.)). Dr. Hu's assertion, of course, assumes that lead from  
 15 baby food *is* absorbed by the body—skirting the question of whether and to what extent the  
 16 beneficial nutrients in food block absorption. Dr. Hu did not investigate this issue. Instead, he opined  
 17 that he was "not aware" of any data demonstrating a difference between food and other exposure  
 18 sources. *Id.* Not only does that answer ignore available data,<sup>7</sup> it also contradicts Dr. Hu's own  
 19 testimony that certain nutrients in food "are well known to have some influence on the absorption  
 20 of lead in food," Kiser Decl., Ex. 4 at 23:16-22 (Hu MDL Tr.), and that "without looking at the  
 21 effect of multiple nutrients, . . . you don't get the full picture" of how nutrients affect the way lead  
 22 is "absorbed from the gastrointestinal tract." *Id.* at 187:1-188:16; *see also id.* at 208:3-9 (discussing  
 23 the "good appreciation of some vitamins having antioxidant effects" because they can "reduce  
 24 oxidative damage or reverse or prevent oxidation."); Kiser Decl., Ex. 34, at 209:17-20 (Aschner  
 25 MDL Tr.) (agreeing that fruits, vegetables, and grains "of course" have antioxidant effects).

26 \_\_\_\_\_  
 27 <sup>7</sup> Ziegler (1979) demonstrates that, for infants and children who consume low levels of lead in food,  
 28 the rate of absorption is negligible and excretion of lead in feces and urine can be greater than the  
 rate of intake, as Dr. Jones admitted. *See* Defs. Opp. to Pltfs. Mot. to Exclude Defs. Expert Witnesses  
 (Dkt. No. 637) at 26.

1       Plaintiffs cannot make food “irrelevant” by their own say so. A reliable methodology  
 2 demands that before their experts offer causation opinions regarding lead and arsenic in Defendants’  
 3 baby food, they consider the science demonstrating the effects of food as a complex mixture. Their  
 4 failure to do so renders their methodology unreliable.

5                   **2. Plaintiffs’ Argument That Their Experts Can Ignore the Known  
 6 Benefits of Healthy Food Demonstrates Their Unreliable and Result-  
 7 Driven Methodology.**

8       Plaintiffs dismissively argue that the fact that baby food is widely accepted to be good for  
 9 pediatric neurodevelopment is also somehow “irrelevant” because lead and arsenic are not.<sup>8</sup>  
 10 Relatedly, they advance an incongruous burden-shifting argument, claiming that if eating fruits,  
 11 vegetables, and grains is not proved to result in *lower* lead or arsenic levels, that makes food  
 12 irrelevant to their experts’ general causation opinions. Opp. at 65-66.

13       It is scientifically unreliable for Plaintiffs’ experts to ignore food’s brain benefits, and  
 14 declare it harmful to the developing brain, without any science to back up their claim. This is  
 15 particularly so because the types of foods at issue are known to provide health benefits *despite the*  
 16 *fact* these foods are also known to contain trace amounts of heavy metals from the growing process.  
 17 Kiser Decl., Ex. 26, at 112:10-25 (Ritz MDL Tr.) (“[W]e know that, you know, you recommend a  
 18 nutritious diet for good neurodevelopment as well as general development in children.”); Kiser  
 19 Decl., Ex. 35, at 337:8-10 (Guilarte MDL Tr.) (agreeing that “a healthy diet is good for the  
 20 developing brain”); Kim Decl., Ex. 1, at 497:4-15 (Shapiro Landon Tr., Vol. II) (“I agree that in  
 21 general, there are nutrients that are important for neurodevelopment, and their presence in food is—  
 22 has a beneficial impact on neurodevelopment.”); Kiser Decl., Ex. 34, at 231:23-232:6 (Aschner  
 23 Landon Tr.) (“[T]here’s many essential nutrients in [fruits, vegetables, and grains] so I would

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24       <sup>8</sup> Plaintiffs claim that Defendants’ position is “akin to arguing that water contaminated with poison  
 25 is safe because water is good for humans.” The analogy is inapt for many reasons. For one, the levels  
 26 of heavy metals in Defendants’ baby food are no different from what one would find in other  
 27 comparable fruits, vegetables and grains sold in the grocery store. Thus, Defendants’ baby food is  
 28 not comparable to an isolated event resulting in “poisoning” of an isolated water source compared  
 to “normal” water. Defs.’ Br. No. 3, at 8, 24. Also, unlike water (which does have some lead and  
 arsenic even under “normal” conditions), the nutrients and other constituents in food indisputably  
 interact with heavy metals to affect their uptake, absorption, distribution, and toxicity. *Id.* at 9-10.

1 presume it's beneficial, absolutely."); *see also* Kiser Decl., Ex. 34, at 123:17-21 (Aschner MDL Tr.)  
 2 acknowledging that fruits, vegetables, and grains all "have some level of lead or arsenic in them");  
 3 Kiser Decl., Ex. 36, at 47:2-48:4 (Shapiro MDL Tr., Vol. I) (recognizing that fruits, vegetables, and  
 4 grains "contain some level of lead and arsenic as a result of growing in the soil").

5 Plaintiffs in response to Defendants reference to public health recommendations that  
 6 children at high risk of lead exposure should eat a diet rich in fruits, vegetables, and grains, Plaintiffs  
 7 cite Desai (2021) and Kordas (2024), arguing that food is irrelevant to lead absorption. Opp. at 65-  
 8 66. But Defendants cited this well-accepted public health guidance because the scientific community  
 9 overwhelmingly recommends that children eat the kinds of foods that make up Defendants' baby  
 10 foods ***notwithstanding*** the presence of trace levels of metals. Whether these foods fully remediate  
 11 lead exposure from other sources, they are not recognized as a driver of increased lead levels.

12 Also, Desai and Kordas, do not support Plaintiffs' position.<sup>9</sup> Those authors did not find that  
 13 the nutrients and other constituents in food "irrelevant." Desai found that "[b]ased on existing  
 14 evidence, a balanced, varied diet continues to be a prudent recommendation for all young children,  
 15 including those with low-level lead exposure." Pltfs. Ex. 82, at 478 (Desai (2021)). In Kordas, the  
 16 authors concluded that food choices did not materially impact children's BLLs, likely because  
 17 "when lead exposure is lower overall, ***diets may neither contribute to nor moderate BLLs.***" Pltfs.  
 18 Ex. 83, at 7-8 (Kordas (2024)) (emphasis added). Nothing Plaintiffs cites supports their theory that  
 19 food is a driver of elevated BLLs in children.

20 Plaintiffs' experts cannot assume (in the face of robust contrary data) that Defendants' baby  
 21 foods elevate lead or arsenic levels or result in a net neurodevelopmental harm—specifically autism  
 22 or ADHD. They must investigate their theory and show it to be scientifically sound at a population  
 23 level. They have not done this.

24  
 25  
 26  
 27 <sup>9</sup> Desai and Kordas are cited by Defendants' experts Dr. Kelleher (discussing both) and Dr.  
 28 Fombonne (citing Desai). *See* Declaration of R. Brent Wisner in Support of Motion to Exclude  
 Certain Testimony from Defendants' Expert Witnesses (Dkt. No. 615-1), Ex. 1 at 45-47 (Kelleher  
 Rep.); Ex. 7, at App. C, at 13 (Fombonne Rep.).

**3. Plaintiffs' Experts' Studies Were Not Designed To Assess and Do Not Assess Whether Baby Food Can Cause Autism or ADHD.**

Plaintiffs claim that their experts did not need to rely on food studies because they “evaluat[ed] whether the nutritional composition of baby food (or food in general) can impact the uptake or adverse neurological effects of heavy metals.” Opp. at 67. But this lawyer-crafted phrasing (repeatedly employed across the experts’ reports) turns the question here on its head. Plaintiffs’ experts did not evaluate whether the nutrients and other constituents found in baby food protect against exposures to heavy metals from baby food. They did not consider whether Defendants’ baby food itself can cause autism or ADHD—which is what matters here. Defs.’ Br. No. 3, at 18.

What Plaintiffs’ experts did was look at individual nutrient-metals interaction studies, mostly conducted in children with high levels of lead or arsenic exposure from environmental sources, and designed to study the therapeutic potential for nutrient supplementation in high-risk populations. Many of the studies do demonstrate significant nutrient-metals interactions resulting in reduced levels of metals in children, and not just in the children who are nutrient deficient (a point Plaintiffs’ experts concede).<sup>10</sup> But, most importantly, none of these studies model what happens to trace levels of lead or arsenic from eating healthy food. These studies do not test to what extent, if any, metals from food are absorbed from the gut of a child eating a realistic diet and then retained in the body (including the brain). And none of the studies examine the impact of such absorption or retention on any potential negative biological mechanism or on the clinical outcome of autism or ADHD. See

<sup>10</sup> See, e.g., Kim Decl., Ex. 2, (Feb. 4, 2022 N.C. Hrg Tr.) at 76:23-27 (“Q. [W]e’re all exposed to metals on a daily basis, and how much we consume of healthy things, like calcium and zinc and iron and selenium, even fiber, all affects how well or not we process metals; correct? A. That’s correct.”); Kiser Decl., Ex. 16, at 32 (Shapiro Rep.) (“[H]armful effects of heavy metal exposure may be offset to some degree by adequate intake of essential nutrients such as calcium, iron, zinc, selenium, and omega-3 fatty acids. In part this may be due to interference with heavy metal absorption.”); Kiser Decl., Ex. 10, at 75-83 (Aschner Rep.) (discussing mitigating effects of iron, calcium, vitamin D, and zinc on lead and arsenic absorption); Kiser Decl., Ex 7, at 90-94 (Gardener Rep.) (“Heavy metals can compete with essential nutrients, such as iron and calcium for binding sites.”); Kiser Decl., Ex. 1, at 25-37 (Ritz Rep.) (describing how nutrients compete with certain heavy metals and “may protect against adverse health effects”); Kiser Decl., Ex. 13. at 33 (Guilarte Rep.) (“Studies dating back several decades suggest an association between iron, zinc, or calcium deficiency and increased lead absorption”); Kiser Decl., Ex. 29, at 205:17-25 (Hu MDL Tr.) (agreeing that certain vitamins can mitigate lead absorption and reduce blood lead levels).

1 Opp. at 64-65. Thus, when Plaintiffs' experts claim that “[t]o a reasonable degree of scientific  
 2 certainty, the literature [] reviewed does not support the conclusion that beneficial nutrients found  
 3 in food, including those found in baby food, can offset or mitigate the uptake and/or toxic  
 4 neurodevelopmental effects of lead [or arsenic],” *see, e.g.*, Kiser Decl., Ex. 1, at 31 (Ritz Rep.), they  
 5 are really engaging in a linguistic sleight of hand (not to mention trying to shift the causation  
 6 burden). In fact, Plaintiffs' experts admit that these papers do not address how metals in food affect  
 7 the neurodevelopment of the child eating that food, and Plaintiffs' lawyers cannot now change that  
 8 testimony or divert the Court's attention from those admissions. *See* Defs.' Br. No. 3, at 11.

9 To defend their experts' flawed literature review, Plaintiffs cherry-pick from each expert's  
 10 testimony to try to bridge the analytical gap between what their experts did and what they needed  
 11 to do in order to provide a methodologically sound opinion. As to Drs. Ritz and Gardener, Plaintiffs  
 12 claim that they considered the specific mechanism of oxidative stress (but not other theorized  
 13 mechanisms) and what, if any, effect nutrients would have on the induction of oxidative stress from  
 14 heavy metals. Opp. at 68. But Dr. Ritz agrees that antioxidants in food may “ameliorate oxidative  
 15 stress.” Kiser Decl., Ex. 26, at 236:15-21 (Ritz MDL Tr.). She even does research demonstrating  
 16 those benefits. *Id.* at 231:25-232:10. Moreover, she engaged in no scientific analysis to determine  
 17 whether eating healthy food causes undue elevations in oxidative stress because of trace levels of  
 18 lead or arsenic—let alone to a level that would induce autism or ADHD. While Plaintiffs cite  
 19 Dr. Ritz's musing as to what she “thinks” might be the effect of nutrients in Defendants' food on  
 20 *possible* heavy metal-induced oxidative stress, they omit the critical last sentence of Dr. Ritz's  
 21 testimony, which changes its meaning: “Even so, they [referring to Defendants' baby foods] are  
 22 more nutritious. There's a sufficient amount of antioxidant that, you know, would actually be  
 23 balancing out the oxidative stress.” *Id.* at 227:23-228:1.

24 Similarly, Dr. Gardener did not study whether Defendants' baby food increases oxidative  
 25 stress at all—let alone to a level capable of causing autism or ADHD—or adversely affects any  
 26 other biological process. Dr. Gardener's research outside the context of litigation reaches the  
 27 opposite conclusion. Defs.' Br. No. 3, at 14, n.7. Plaintiffs cite Dr. Gardener's testimony that she  
 28 need not consider food because her charge focused on heavy metals, and so she treated baby food

1 as irrelevant: “[M]y report has to do with heavy metals. And so the impacts of -- of these foods  
 2 independent of heavy metals is beyond the scope of -- of my report.” Kiser Decl., Ex. 46, at 145:19-  
 3 22 (Gardener Landon Tr.). There is no scientifically sound basis for disaggregating food from itself,  
 4 *see* Defs.’ Br. No. 3, at 10-16—children eat Defendants’ baby foods as food, and the failure of  
 5 Dr. Gardener to consider the impact of heavy metals in the context of the whole food children eat is  
 6 a fundamental methodological flaw.<sup>11</sup>

7 Plaintiffs’ arguments as to Drs. Hu, Aschner, Guilarte, and Shapiro fail for similar reasons.  
 8 They all addressed the same question discussed above—whether certain individual nutrients affect  
 9 children exposed to high levels of environmental lead or arsenic—not what happens when children  
 10 eat food. In response to Defendants’ critique that their experts only looked at single nutrient studies,  
 11 Plaintiffs cite to the experts *looking at single nutrient studies*. Opp. at 69-70. For example, despite  
 12 Plaintiffs’ claim that Dr. Aschner’s “focused almost 40 pages of his report on the issue of metal  
 13 exposure through baby food and the interaction between the nutritional composition of the foods  
 14 and heavy metals,” *id.* at 70, Dr. Aschner merely looked at individual nutrient-interaction studies—  
 15 not studies assessing the complex interactions that occur when children eat food like Defendants’  
 16 baby foods. *See* Kiser Decl., Ex. 10, at 72-93 (Aschner MDL Rep.). Plaintiffs admit that Dr. Guilarte  
 17 did not examine “whole food,” because he does not “think that study has been done.” Opp. at 71  
 18 (quoting Kiser Decl., Ex. 35, at 364:6-19 (Guilarte MDL Tr.)). Indeed, Plaintiffs do not rebut the  
 19 testimony Defendants cited, in which all of Plaintiffs’ experts admit that they failed to consider food  
 20 as a whole complex mixture. *See* Defs.’ Br. No. 3, at 10-15.

21 Plaintiffs’ experts rely on nutrient-metal studies that ask and answer the wrong question—  
 22 how nutrients affect children at high risk of lead or arsenic exposure *not* from food. Plaintiffs’  
 23 experts cannot reliably extrapolate from those studies to draw conclusions about baby food.

24

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25 <sup>11</sup> Plaintiffs argue that Dr. Gardener devoted “[n]o fewer than 30 pages” of her report to “evaluating  
 26 the issue of whether the nutritional composition of baby food (or food in general) affects the uptake  
 27 or adverse neurological effects of metals.” Opp. at 69. Again, this is the same artful phrasing  
 28 Plaintiffs’ lawyers used above—she reviewed whether individual nutrients therapeutically  
 remediate high dose metals exposures generally. She did not investigate food as a whole complex  
 mixture or what happens when children eat food. *See id.* (acknowledging that Dr. Gardener focused  
 on “constituent nutrients” only).

**4. Plaintiffs' Defense of Their Experts' Failure to Cite to Studies Showing No Increase in Autism or ADHD Tied to Eating Healthy Foods Further Reveals Their Unreliable and Result-Oriented Methodology**

Finally, Plaintiffs try to dismiss as “not informative” studies that Defendants’ argued should have been considered as part of a sound methodology because they evaluate whether diet impacts autism risk. Plaintiffs’ Opposition only underscores their experts’ inconsistent and result-driven approach. Opp. at 71-72.

Plaintiffs’ summary dismissal of food studies as “not particularly informative of the issues in this litigation,” underscores their experts’ results-driven approach. *Id.* Plaintiffs point to Dr. Ritz’s testimony that certain food studies are not relevant because of their cross-sectional design, which does not satisfy temporality. *Id.* (discussing Rahbar (2021) and Rahbar (2022)); *see* Defs.’ Br. No. 3 at 12-13. Yet Dr. Ritz’s causation opinions, like those of all Plaintiffs’ experts, rest almost entirely on cross-sectional or case control studies that fail temporality. In fact, Plaintiffs’ Opposition repeatedly argues that cross-sectional studies *not involving food* prove cause and effect from food. *See* Opp. at 64-67, 71-73, 93-97.

Plaintiffs also quote Dr. Ritz’s dismissal of studies showing an association between earlier introduction of solid foods and a *lower* risk of autism. *See* Kiser Decl., Ex. 26, at 132:14-134:24, 275:8-277:22 (Ritz MDL Tr.) (dismissing Xiang (2023) and Emond (2010)); *see also* Kiser Decl., Ex. 62 (Emond (2010)); Kiser Decl., Ex. 63 (Campbell (2024)); Kiser Decl., Ex. 79 (Xiang (2023)). Dr. Ritz testified that these papers could reflect “reverse causation”—the children could have already been autistic at the point they began eating food and the presence of autism could have affected food selection rather than the other way around. *See, e.g.*, Kiser Decl., Ex. 26, at 134:1-6 (Ritz MDL Tr.) (asserting that a child’s delayed consumption of solid foods could be “reverse causation” “[b]ecause a child that’s already on the spectrum might be fussy and not the other way around”). Plaintiffs also quote Dr. Aschner’s testimony that the Rabar data were not instructive because they did not measure the amount of lead or arsenic in any foods the children consumed.

But Dr. Ritz's (and all of Plaintiffs' experts') causation opinions fundamentally depend upon the unproven assumption that children do not already have autism at the time they start eating Defendants' baby foods. Further, none of the papers they cite measure the amount of exposure from

1 any source—let alone from food. They only measure body levels of lead or arsenic, typically without  
 2 knowing how or in what amounts children were exposed. Dr. Ritz and Plaintiffs' other causation  
 3 experts cannot explain away their failure to grapple with these data *in food*, which are inconsistent  
 4 with their opinions, while firmly resting their food causation opinions on non-food data that present  
 5 the same (indeed, far greater) limitations. That inconsistency demonstrates the results-oriented and  
 6 unreliable nature of Plaintiffs' experts' opinions.

7 In short, the fundamental absence of evidence that food consumption can cause autism or  
 8 ADHD is fatal to Plaintiffs' claim. Plaintiffs' expert's attempted workarounds do not bridge the  
 9 analytical gaps and leaps of logic that mandate dismissal under Rule 702.

10 **B. Plaintiffs' Resort to an "Any Dose, Any Exposure, Any Window" Approach Is  
 11 Improper and Cannot Satisfy Plaintiffs' Obligation to Provide Reliable  
 12 Evidence of General Causation.**

13 Plaintiffs claim that their experts do address the dose of lead or arsenic from baby foods and  
 14 rely on four studies to support their extrapolation from the literature to Dr. Jones' estimated doses.  
 15 *See Opp. at 76-83.* They also argue that, in the absence of a known "safe dose," whether a particular  
 16 dose will cause autism or ADHD is entirely dependent on the child. Opp. at 83-84. In other words,  
 17 they claim that they can skip past general causation because any dose can cause autism or ADHD,  
 18 depending on a child's alleged "vulnerabilities" (which they cannot define or measure). Plaintiffs'  
 19 first argument is inconsistent with the record, and their second argument is inconsistent with their  
 legal burden under Rule 702 to establish general causation.

20 **1. Plaintiffs' Experts Do Not Engage in Any Reliable Methodology to  
 21 Connect Dr. Jones' "Doses" to the Scientific Literature.**

22 Plaintiffs claim that their experts rely on sufficient evidence of "augmented risk for  
 23 developing ASD/ADHD following exposure to specific doses of heavy metals." Opp. at 77. But  
 24 Plaintiffs' experts do not scientifically link Dr. Jones' calculations to the general heavy metal  
 25 literature, nor do the four papers cited by Plaintiffs provide a reliable foundation to do so.

26 **a) Plaintiffs' Experts Do Not Link Dr. Jones' Calculations to the  
 27 General Heavy Metal Literature.**

28 Contrary to Plaintiffs' claim (Opp. at 77-79), none of Plaintiffs' causation experts seriously

1 assessed Dr. Jones' dose calculations or compared them to any epidemiological literature. Dr. Ritz  
 2 admitted she did not "compare" Dr. Jones' calculations to the literature, Defs.' Br. No. 3, at 23, and  
 3 Dr. Guilarte did not even review Dr. Jones' exposure calculations before finalizing his own report  
 4 purporting to rely on Dr. Jones. *See* Kiser Decl., Ex. 35, 16:13-17:14 (Guilarte MDL Tr.). Dr. Hu  
 5 likewise admitted that he does not have an opinion on what change in blood lead level is "meaningful  
 6 or significant" in terms of autism or ADHD risk. Defs.' Br. No. 3, at 23.<sup>12</sup> Plaintiffs instead argue  
 7 that Dr. Ritz believes she "can calculate" a dose from the literature, and that Dr. Hu believes the  
 8 literature is "simply straightforward" in confirming that exposure at the levels encountered in baby  
 9 food "is likely to be a risk factor for autism." Opp. at 81-82 (quoting Kiser Decl., Ex. 29, at 83:3-9,  
 10 199:22-200:11, 233:7-234:2 (Hu MDL Tr.) and Kiser Decl., Ex. 26, at 163:2-10 (Ritz MDL Tr.)).  
 11 But Dr. Ritz *did not* perform any such calculations and has proffered no methodology for doing so,  
 12 and Dr. Hu's statements beg the question at the heart of the general cause proceeding as to what the  
 13 studies show. His opinions are nothing more than speculation and his own say-so.

14 While Dr. Gardener references Dr. Jones' outputs, she does not connect them to any studies  
 15 or rely on them for her general causation opinion. Rather, she resorts to a "no safe level" opinion:  
 16 "in the literature, there is no level of lead exposure below which we can say, you know, autism or  
 17 ADHD would not be causally related." Kiser Decl., Ex. 33, at 245:11-24 (Gardener MDL Tr.); *see*  
 18 *also id.* at 247:3-8 ("There is no amount that is minimally—there is no minimum amount of exposure  
 19 or—below which autism—lead and arsenic don't cause autism and ADHD, nor is there a maximum  
 20 amount. I mean, there's an amount a child might die, but they might die not autistic, without  
 21 ADHD.") The same is true of Dr. Aschner, who could not identify "any set of values that Dr. Jones  
 22 could have presented" where he would not have been able to opine that lead or arsenic exposure  
 23 was capable of "resulting in symptoms that might be a part of the diagnosis of autism or ... ADHD."  
 24 Kiser Decl., Ex. 34, at 226:1-19 (Aschner MDL Tr.) If Drs. Gardener and Aschner are simply  
 25

26 <sup>12</sup> Dr. Shapiro, who Plaintiffs now claim is limiting his opinions to the biological plausibility of food  
 27 consumption causing autism/ADHD, did not consider dose *at all* as a general causation question.  
 28 Opp. at 81, n. 27; *see* Kiser Decl., Ex. 36, at 14:23-15:4 (Shapiro MDL Tr., Vol. I) ("So I have not  
 considered the specific doses of intake that might be relevant in a particular individual with those  
 diagnoses.").

1 offering “any dose” opinions, then Plaintiffs can hardly claim that they have reliably linked  
2 Dr. Jones’ calculations to the epidemiological literature.

3 In short, Plaintiffs' own experts' testimony belies Plaintiffs' claim that their experts engaged  
4 in a scientifically rigorous analysis to conclude from the epidemiological literature that children  
5 exposed to heavy metals through baby food at the levels Dr. Jones estimates are at an increased risk  
6 of autism and ADHD. Plaintiffs' experts, at most, retreated to their "no safe dose" opinions, and at  
7 worst, relied on ***no foundation*** to bridge the gap between the epidemiology and Dr. Jones' purported  
8 dose calculations. This failure is fatal to their opinions. *GE v. Joiner*, 522 U.S. 136, 144 (1997)  
9 (finding a proponent's failure to explain why their experts could reliably extrapolate from certain  
10 studies fatal to the experts' opinions); *Schudel v. GE*, 120 F.3d 991, 997 (9th Cir. 1997) (excluding  
11 expert because there was no showing that extrapolation was scientifically acceptable); *In re Bextra*  
12 and *Celebrex Mktg. Sales Pracs. & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1180 (N.D. Cal. 2007)  
13 ("*Bextra*") (holding that an expert is required to provide evidentiary support "that suggests that their  
14 experts' extrapolation is scientifically valid").

**b) The Literature Plaintiffs Now Cite Does Not Provide a Reliable Foundation for Their Experts' Causation Opinions.**

17 Plaintiffs cite four studies that they argue reveal associations between autism or ADHD and  
18 the lead or arsenic levels Dr. Jones estimated. *See* Opp. at 76-80. None of those studies does anything  
19 of the sort.

20 Two studies, Ha (2008) and Kim (2016), constitute Plaintiffs' examples of epidemiological  
21 literature with lead exposure levels against which they claim Dr. Jones' estimates can be compared.  
22 Opp. at 77-78. Ha (2008) measured blood lead and mercury levels in Korean children aged 6-10 and  
23 reported that those with BLLs higher than 1 ug/dL were more likely to register a certain score on an  
24 abbreviated Korean ADHD screening tool. Pltfs. Ex. 85, at 33-36 (Ha 2008). But nearly all of Dr.  
25 Jones' mean BLL are *below* 1 ug/dL. *See, e.g.*, Kiser Decl., Ex. 21, at Table 33 (Jones Rep.); Kiser  
26 Decl., Ex. 24, at Tables 7, 10, 16, 21, 22, 28 (Jones Amended Rebuttal Rep.). And Dr. Jones'  
27 exposure window is tied to when children eat baby food—decidedly earlier than 6-10 years old.  
28 Importantly, the Ha authors advised caution in finding causation from their study results because

1 the study employed a cross-sectional design (thus not satisfying temporality) and did not use an  
 2 accepted diagnostic endpoint. Pltfs. Ex. 85, at 35-36 (Ha 2008). As they noted, the study “could not  
 3 address the relationship between lead exposure earlier in life and ADHD” because the blood  
 4 “concentrations were performed at the time of assessing the ADHD symptoms.” *Id.* Also, the ADHD  
 5 screening assessments they studied “were not obtained using a rigorous protocol.” *Id.* The symptom  
 6 scale they employed is typically used only as a “screening tool before definitive diagnostic  
 7 procedures.” *Id.* Finally, ***none of the study findings were statistically significant***, meaning that the  
 8 study’s conclusions were just as likely to be the result of chance as they are a true association.

9 Kim (2016) studied autism-related symptoms in Korean children at ages 7-8 years, 9-10  
 10 years, and 11-12 years, but ***excluded children with an autism diagnosis***. Kiser Decl., Ex. 85 at 194  
 11 (Kim 2016). The authors reported that a “one-unit increment” in BLL at 7-8 years of age was  
 12 associated with a minor increase (0.151; 95% CI: 0.061, 0.242) in reported autism symptoms at 11-  
 13 12 years of age, but blood lead levels in those same children taken at ages 9-10 and 11-12 were ***not***  
 14 ***associated*** with reported autism symptoms at 11-12 years of age. *Id.* at 196. The authors do not  
 15 identify at what doses excess risk in the 7-8 year old group became apparent, only identifying the  
 16 mean BLL of 1.64 ug/dL in the entire cohort. *Id.* at 196-197 (Tables S1, S2).

17 There is no relationship between these data and Dr. Jones’ estimates. Most blatantly, the  
 18 Kim study gives *no numerical “dose”* of lead exposure to which Dr. Jones’ doses could be compared  
 19 without further statistical analysis. *Id. passim*. Plaintiffs’ experts never explain how a 0.5 ug/dL  
 20 BLL in Dr. Jones’ calculations, for example, compares to a “one unit increment” in BLL in the Kim  
 21 study. *See* Opp. at 77-78. And all but two of the 21 mean BLLs Dr. Jones calculated using the  
 22 hypothetical menus she was provided fall *below* that mean BLL in Kim. Kiser Decl., Ex. 21, at  
 23 Table 33 (Jones Rep.); Kiser Decl., Ex. 24, at Tables 7, 10, 16, 21, 22, 28 (Jones Amended Rebuttal  
 24 Rep.). Further, given that an association was reported only with increased BLLs measured in *one of*  
 25 *three age groups* (7-8 year-olds), there is no reliable method to compare these results with the BLLs  
 26 that Dr. Jones estimated for much younger children. In Kim, the age at the time of measuring BLLs  
 27 mattered, but the study did not assess BLLs at the ages relevant to this litigation. *See* Kiser Decl.,  
 28 Ex. 85 at 194 (Kim 2016). Finally, the Kim study excluded children with autism, and Plaintiffs’

1 experts give no explanation for why they can draw conclusions about autism from a study that  
 2 involves ***no autistic children***.<sup>13</sup> As even the authors acknowledge, “clinical ASD and autistic  
 3 behaviors evaluated using tools such as the ASSQ or SRS are not the same[.]” Kiser Decl., Ex. 85,  
 4 at 198 (Kim 2016).

5 The arsenic studies Plaintiffs cite, Zhu (2024) and Tsuji (2025), similarly fail to reliably  
 6 support their experts’ conclusions. Opp. at 79-80. Only two of Plaintiffs’ experts, Drs. Aschner and  
 7 Shapiro, cite Zhu, and neither connects any “dose” of arsenic from Zhu to the estimated doses  
 8 generated by Dr. Jones. *See* Opp. at 81-83; *see also* Kiser Decl., Ex. 10, at 24 (Aschner Rep.); Kiser  
 9 Decl., Ex. 16, at 27 (Shapiro Rep.). And for good reason: Zhu looked at “autism-like changes” in  
 10 zebrafish swimming in water with high concentrations of arsenic. Pltfs. Ex. 88, at 12 (Zhu (2024)).  
 11 Not only is it patently unscientific to extrapolate “autism-like changes” in swimming zebrafish to  
 12 actual neurological disorders in human children eating baby food, but the study also provides no  
 13 “dose” that Plaintiffs’ experts could compare to Dr. Jones’ estimates. *See generally id.*

14 Finally, in relying on Tsuji (2015), Plaintiffs ignore the authors’ conclusion that “the overall  
 15 evidence **does not consistently show a causal dose-response relationship at low doses**[.]” *See* Opp.  
 16 at 82-83; Pltfs. Ex. 87, at 91 (Tsuji (2015)) (emphasis added). Plaintiffs’ experts rely on the study’s  
 17 suggestion of a “possible” association between arsenic and *cognitive function*, based on Bangladesh  
 18 studies, and suggested regulatory reference dose of 0.0004-0.001 mg/kg-day—assuming the “most  
 19 rigorous evidence from Bangladesh is generalizable to U.S. populations.” Pltfs. Ex. 87, at 91 (Tsuji  
 20 (2015)). The Tsuji study does not hypothesize any dose of arsenic that could be associated with  
 21 autism, and even with cognitive function, the “overall evidence” does not consistently show a causal  
 22 relationship at low doses. *Id.* at 96. In light of the study’s conclusion that the totality of the evidence  
 23 does not support a causal association between arsenic exposure and any endpoint, much less autism,  
 24 Plaintiffs’ experts cannot reliably conclude from this study that children with arsenic exposures at  
 25

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26 <sup>13</sup> At the time of writing his report, Dr. Hu did not even know that the Kim study excluded children  
 27 with autism. Kiser Decl., Ex. 29, at 267:10-271:5 (Hu MDL Tr.) (“Q. Have you looked before today  
 28 as to whether children were—with autism were excluded from the Kim study? [objection] The  
 Witness: I haven’t, but I—***it just would make no sense why they would exclude children with  
 actual autism***. That would make absolutely no sense[.]”) (emphasis added).

1 the levels Dr. Jones estimates are somehow at higher risk for autism.<sup>14</sup>

2 Plaintiffs' experts never compared the literature to Dr. Jones' calculations. But even if they  
 3 had, the four studies Plaintiffs' lawyers now say bridge the analytical gap do not. There is no reliable  
 4 basis to extrapolate from Plaintiffs' supposed dose-relevant studies to Plaintiffs' claim that  
 5 consuming Defendants' baby foods can cause autism or ADHD. Defs.' Br. No. 3, at 21-25.

6 **2. Plaintiffs' Experts' "Any Dose" Opinions Are Insufficient to Establish  
 7 General Causation.**

8 Finally, Plaintiffs argue that their experts are not giving an "any dose" opinion, but rather,  
 9 an "any dose, depending on the child" opinion. *See* Opp. at 83-84. This is a distinction without a  
 10 difference, as neither can reliably establish general causation.

11 Because the general heavy metal epidemiological literature cannot be reliably compared to  
 12 Dr. Jones' exposure estimates, Plaintiffs' experts ultimately fall back on their "no safe dose" theory  
 13 to extrapolate from one to the other. *See e.g.*, Kiser Decl., Ex. 33, 245:11-24 (Gardener MDL Tr.);  
 14 Kiser Decl., Ex. 26, at 180:21-181:9 (Ritz MDL Tr.) (explaining that she did not need to compare  
 15 Dr. Jones' dose estimates to ranges in the literature because "there's no safe level, so we're allowed  
 16 to extrapolate"); Kiser Decl., Ex. 29, at 83:3-9 (Hu MDL Tr.) ("There is no threshold that I'm aware  
 17 of that's been identified below which, you know, lead could not be expected to have a deleterious  
 18 effect.").

19 Recognizing the methodological failings of that approach, Plaintiffs argue that their experts  
 20 "do not opine that **any** level of exposure **will** cause ASD/ADHD," but rather that, "while any amount  
 21 of exposure confers a risk on a population level, the dose makes the poison **depending on the**  
 22 **individual.**" Opp. at 84 (emphasis in original). But the methodological question Defendants identify

23  
 24 <sup>14</sup> Unable to link Dr. Jones' doses to the epidemiology, Plaintiffs resort to regulatory reference doses  
 25 for lead. Opp. at 78. Plaintiffs argue that the FDA's interim reference level ("IRL") for lead exposure  
 26 is 2.2 ug/day, and consuming the hypothetical menus of each Defendant's baby foods that Plaintiffs'  
 27 counsel constructed could exceed this level. *Id.* But notably, Plaintiffs omit that the FDA clarified that  
 28 its regulatory reference dose for lead "includes an additional 10x safety factor[.]" Plts. Ex. 15, at 5-6  
 (Action Levels for Lead in Processed Food Intended for Babies and Young Children: Guidance for  
 Industry). And the IRL is not tied to any autism or ADHD outcome. As such, the reference level  
 cannot be used to compare to Dr. Jones' lead exposure estimates or to establish causation.

1 is that Plaintiffs' experts are in fact claiming that *any amount of exposure* can cause autism or  
 2 ADHD, without addressing what amount of increased risk is sufficient to *cause* autism and ADHD  
 3 at a population level. General causation does not address any particular child. It asks a different  
 4 question: whether the population of children exposed to a "realistic" dose of lead or arsenic from  
 5 consuming Defendants' baby foods is statistically significantly more likely to develop autism or  
 6 ADHD than the population of children exposed to a lower dose of those heavy metals through  
 7 alternative food choices. *See, e.g., McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1241 (11th Cir.  
 8 2005) (explaining that "a plaintiff must demonstrate the levels of exposure that are hazardous to  
 9 human beings generally" (internal quotations omitted)); *Mitchell v. Gencorp Inc.*, 165 F.3d 778, 781  
 10 (10th Cir. 1999) (same); *Wright v. Willamette Indus.*, 91 F.3d 1105, 1106 (8th Cir. 1996) (same);  
 11 *Allen v. Penn. Eng'g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996) ("Scientific knowledge of the harmful  
 12 level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities,  
 13 are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case."). Plaintiffs skip  
 14 this step entirely and their experts presume that in a hypothetical "susceptible" child eating even a  
 15 single carrot with any amount of lead could cause autism or ADHD. Kiser Decl., Ex. 36, at 151:20-  
 16 152:4 (Shapiro MDL Tr., Vol. I). Plaintiffs' experts concede they have no way of determining which  
 17 children are "susceptible," meaning that in any particular child, the fact that the child developed  
 18 autism or ADHD is, in and of itself, evidence that the child was susceptible. Kiser Decl., Ex. 36, at  
 19 70:24-71:24 (Shapiro MDL Tr., Vol. I). This circular reasoning cannot support a reliable method.

20 In each case in this MDL, Plaintiffs must show both general and specific causation—that is,  
 21 that on a population level, consuming Defendants' baby foods presents an increased risk of autism  
 22 or ADHD *and* that a particular child's autism or ADHD was caused by that consumption. Plaintiffs  
 23 cannot use the latter to prove the former. By defaulting to an "any dose, depending on the individual"  
 24 opinion, Plaintiffs attempt to skip the relevant general causation inquiry.

25 **C. Plaintiffs Fail to Explain How the Non-Food Studies Their Experts Rely Upon  
 26 Reliably Support Opinions Regarding Whether Defendants' Baby Foods Can  
 Cause Autism or ADHD.**

27 Plaintiffs' extrapolations from non-food studies to establish general causation as to baby  
 28 food products are also inherently methodologically flawed. *See* Defs.' Br. No. 3, at 25-39. Even

1 setting aside that Plaintiffs do not meaningfully address the threshold problem that almost 90% of  
 2 their studies do not satisfy temporality, Plaintiffs provide no scientifically acceptable justification  
 3 for their experts extrapolating from studies examining (1) mere symptoms, as opposed to diagnosis,  
 4 of autism or ADHD—especially when these symptoms are common to many conditions; (2) prenatal  
 5 data for lead and arsenic exposure; or (3) data from non-U.S. populations. Instead, Plaintiffs,  
 6 viewing general causation as an exercise in study-counting, accuse Defendants of trying to “erase  
 7 the overwhelming body of scientific data clearly linking lead and arsenic exposure to ASD and/or  
 8 ADHD,” and point to “hundreds of published epidemiological studies supporting Plaintiffs’ experts’  
 9 general causation opinions,” Opp. at 84-85. Plaintiffs’ experts rely on scientific studies that do not  
 10 address the relevant inquiry, which is ***whether each Defendant’s baby food products can cause***  
 11 ***ASD or ADHD***—not, as Plaintiffs describe it, “the ability of lead and arsenic to increase the risk of  
 12 ASD and/or ADHD in children.” Opp. at 85. Plaintiffs offer no legitimate reason for this Court to  
 13 accept the extrapolations that their experts proffer.

14           1.     **Plaintiffs Fail to Justify Their Experts’ Improper Reliance on**  
 15           **Literature that Does Not Study Diagnosed Autism or ADHD as an**  
 16           **Endpoint.**

17           Plaintiffs do not, and cannot, dispute that their experts rely on literature that fails to assess  
 18 the specific injuries alleged—diagnosed autism and ADHD. Defs.’ Br. No. 3, at 27-31; Opp. at 85-  
 19 88. Here again, Plaintiffs’ Opposition is remarkable for what it does *not* say. Defendants explained  
 20 why it is methodologically improper for Plaintiffs’ experts to draw conclusions about autism and  
 21 ADHD from studies that evaluated only symptoms such as whether a child is clumsy, bullied, or an  
 22 “eccentric professor.” Defs.’ Br. No. 3, at 29. In response, Plaintiffs fail to cite any textbook, treatise,  
 23 or other scientific authority demonstrating that their experts can reliably extrapolate from symptoms,  
 24 behaviors, and screening questionnaires to conclusions about autism and ADHD. *See* Opp. at 86-  
 25 87. Plaintiffs’ experts proffer no data demonstrating that any of the specific screening scales used  
 26 to assess symptoms or behaviors in the studies they rely upon are used to make a proper diagnosis  
 27 by a qualified medical professional. Kiser Decl., Ex. 4, at 33 (Hu Rep.); Kiser Decl., Ex. 13, at 18-  
 28 24, 27-32 (Guilarte Rep.); Kiser Decl., Ex. 10, at 26-29, 34-47 (Aschner Rep.). Plaintiffs also do not  
 dispute that screening scales and questionnaires capture symptoms or behaviors that are common in

1 children who do not have autism or ADHD. *See, e.g.*, Defs.’ Br. No. 3, at 29-30. Plaintiffs never  
 2 address this point or the authors’ acknowledgements in the studies they rely upon which make clear  
 3 the symptoms or traits subjectively assessed are not the same as diagnosed autism or ADHD.

4 Instead, Plaintiffs make three arguments: (1) “symptoms” are a more useful endpoint than  
 5 diagnosed autism or ADHD because they capture the “degree[s] of impairment” within the autism  
 6 spectrum (Opp. at 86); (2) screening tools are strongly associated with autism and ADHD (*id.* at 86-  
 7 87); and (3) studies of “symptoms” strengthens the confidence of their opinions (*id.* at 87-88).<sup>15</sup>  
 8 None of these arguments, which largely rely on Plaintiffs’ experts’ *ipse dixit*, justifies reliance on  
 9 studies with symptom-only endpoints to support their general causation opinions.

10 As to the first argument, no Plaintiff in this MDL is seeking redress for a “degree of  
 11 impairment” within the spectrum of these behavioral disorders *short of actual, diagnosed autism*  
 12 *and ADHD*. *See* Opp. at 86. Whether studies of non-specific behaviors that may or may not be  
 13 associated with autism and/or ADHD are “useful” for some purpose is immaterial; those studies  
 14 cannot support a reliable causation opinion about autism or ADHD. At least some of Plaintiffs’ own  
 15 experts agree: Dr. Guilarte admitted that “***to reach reliable opinions about causation of ASD, you***  
 16 ***need to look at diagnosed ASD***” and “***the same is true for ADHD***.” Kiser Decl., Ex. 35, at 71:18-  
 17 72:4 (Guilarte MDL Tr.) (emphasis added). Dr. Shapiro similarly acknowledged that a positive  
 18 result on a screening scale does not equate with an actual diagnosis, that autism is “a clinical  
 19 diagnosis,” and the symptoms or traits, such as reductions in intelligence, hyperactivity, and  
 20 behavioral problems, are not specific to autism. *See* Defs.’ Br. No. 3, at 30. And Dr. Hu admitted  
 21 that decreased IQ—one of the “symptoms” measured in the studies—is not even part of the criteria  
 22 for an autism diagnosis. *Id.* at 28 (citing Kiser Decl., Ex. 29, at 154:13-18 (Hu MDL Tr.)). Plaintiffs’  
 23 experts cannot treat symptoms and diagnoses the same for purposes of offering a causation opinion,  
 24 while conceding that the two are not the same at all.

25 Plaintiffs next suggest that the authors of the studies on which their experts rely confirmed  
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27 <sup>15</sup> Plaintiffs also claim they did not rely solely on literature assessing only symptoms. As described  
 28 more fully in Section II.D.2, *infra*, the scant literature involving diagnosed autism and ADHD  
 cannot be used to support a reliable causation opinion for other reasons.

1 that screening tools, such as ASSQ and SRS, are “strong[ly] associate[ed]” with diagnosed autism  
 2 and ADHD. Opp. at 86-87. As discussed *supra*, studies cited by Plaintiffs’ experts say the opposite.  
 3 For example, the Kim (2016) authors acknowledge that “clinical ASD and autistic behaviors  
 4 evaluated using tools such as the ASSQ or SRS are not the same[.]” Kiser Decl., Ex. 85, at 198 (Kim  
 5 2016); *see also* Pltfs. Ex. 85, at 36 (Ha 2008). Sioen (2013) similarly provides that “the SDQ  
 6 hyperactivity score cannot be considered similar as a ADHD diagnosis.” Kim Decl., Ex. 3, at 230  
 7 (Sioen 2013) (cited by Dr. Hu, Kiser Decl., Ex. 4, at 13 (Hu Report)). And Sciarillo (1992) explains  
 8 that “[t]he [CBCL] subscales represent factor analytically derived behavioral descriptions, not  
 9 diagnostic inferences.” Kim Decl., Ex. 4, at 1358 (Sciarillo 1992) (cited by Dr. Ritz, Kiser Decl.,  
 10 Ex. 1, at 63 (Ritz Report)). The DSM-5 confirms this key distinction between diagnoses and  
 11 symptoms, explaining for autism: “the diagnosis remains a clinical one, taking all available  
 12 information into account,” and it “is not solely dictated by the score on a particular questionnaire or  
 13 observation measure.” American Psychiatric Association, Diagnostic and Statistical Manual of  
 14 Mental Disorders DSM-5-TR, 62 (5th ed. 2022), [Diagnostic and Statistical Manual of Mental](#)  
 15 [Disorders, Fifth Edition, Text Revision \(DSM-5-TR\(tm\)\)](#).<sup>16</sup>

16 Finally, Plaintiffs argue that using screening tools to find symptoms of autism and ADHD  
 17 somehow strengthens their conclusions about diagnosed autism and ADHD. Opp. at 87-88. That is,  
 18 Plaintiffs dodge Defendants’ argument entirely: nowhere do Plaintiffs’ experts proffer any data that  
 19 the specific screening scales that assess symptoms or behaviors can serve as a reliable proxy for an  
 20 actual diagnosis.

21 Ninth Circuit law “demands a showing of reliability” and evidentiary support “that suggests  
 22 that [plaintiffs’] experts’ extrapolation is scientifically valid.” *In re Bextra*, 524 F. Supp. 2d at 1180  
 23

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24 <sup>16</sup> Nor do Plaintiffs’ ever address that none of these studies assess key criteria for an autism diagnosis.  
 25 A hallmark of social-communicative deficits that define autism is the presence of impairment in the  
 26 use of gestures and non-verbal communication (criterion A2). DSM-5-TR, *supra*, at 56. Yet, no study  
 27 of heavy metal exposure shows detrimental effects on non-verbal communication. Nor have any  
 28 studies on symptoms or traits examined the lack of social reciprocity (criteria A1 and A3) that is core  
 to autism and necessary for the diagnosis. *Id.* Finally, no study of heavy metal exposure has shown  
 detrimental effects on repetitive and ritualized behaviors laid out in criterion B. *Id.* at 56-57. Plaintiffs’  
 experts do not confront these fundamental limitations in the studies that use symptoms or traits from  
 screening scales instead of an actual autism diagnosis.

1 (excluding opinion where expert “failed to identify any scientific support for his method other than  
2 his own judgment.”); *see also In re Acetaminophen – ASD-ADHD Prods. Liab. Litig.*, 707 F. Supp.  
3 3d 309, 364 (S.D.N.Y. 2023) (excluding general causation experts who failed to “confine  
4 themselves to studies that relate to diagnoses of ASD and ADHD” and relied on “studies of  
5 symptoms that reflect many endpoints relevant to [neurodevelopmental disorders] generally,  
6 including to ASD and ADHD.”).<sup>17</sup> Nothing other than Plaintiffs’ experts’ say-so supports their  
7 opinion that studies of symptoms, behaviors, or traits can reliably be used to offer opinions about  
8 clinically diagnosed autism or ADHD—let alone “more useful” than studies involving a diagnosis.  
9 Opp. at 86 (quoting Dr. Shapiro). Because too great a gap exists between the studies on symptoms  
10 and the opinions offered by Plaintiffs’ experts, this Court should exclude them.

**2. Plaintiffs Fail to Justify Their Experts' Reliance on Studies from the Prenatal Period or from the Post-Natal Period After the Window for Consumption of Baby Food.**

13 Defendants' motion argued that studies of prenatal exposure to lead or arsenic, and postnatal  
14 studies from the period after children consume baby food, cannot reliably support Plaintiffs' experts'  
15 general causation opinions. Defs.' Br. No. 3, at 43-47. In response, Plaintiffs focus exclusively on  
16 attempting to rebut arguments regarding prenatal studies. *See* Opp. at 91. Plaintiffs have no response  
17 as to studies examining the post-baby food consumption period. As to the prenatal studies, Plaintiffs'  
18 arguments fall into two equally unavailing buckets: that prenatal studies are useful for other  
19 purposes, such as considerations of biological plausibility and reverse causation, and that Plaintiffs'  
20 experts can reliably extrapolate from the prenatal to postnatal period.<sup>18</sup>

22 <sup>17</sup> Plaintiffs' reliance on N.C., 2022 WL 21778549 (2023), is also unavailing. That decision, based  
23 on a different standard in California state court, also assessed a different causation question than the  
24 one in this case, as this Court has already recognized. See Kiser Decl., Ex. 55, at 39:23-25  
25 (2024.06.20 CMC Tr.). The N.C. court determined that “[f]rom the Court's point of view, the extent  
26 to which a study is 'unreliable' is a matter properly reserved for cross-examination at trial.” N.C. v.  
27 *Hain Celestial Group, Inc.*, No. 21STCV22822, 2023 WL 8261722, at \*12 (Cal. Super. Sept. 1,  
2023). Nonetheless, the court ultimately excluded Plaintiff's exposure and specific causation experts  
for failing to adequately apply the general heavy metals literature to the relevant causation  
question—whether *baby food* caused the plaintiff's autism. *Id.*

<sup>27</sup> Plaintiffs also take issue with Defendants' argument that prenatal studies do not support causation, citing five studies in response. Defendants address those studies in Section II.D.2, *infra*, and here, focus on the arguments relating to extrapolation from prenatal studies.

1       As to the first argument, the question is not whether prenatal studies may be useful for some  
 2 other purpose. It is whether they are informative to the general causation question here. They are  
 3 not, because they do not implicate the developmental window at issue here.

4       As to the second argument, citing only their own experts' say-so, Plaintiffs claim that  
 5 prenatal studies support their postnatal causation opinions because "[b]rain development is  
 6 protracted and continuous and 'occurs during the prenatal period' and 'continues on after' for 'years  
 7 and years.'" Opp. at 92 (quoting Kiser Decl., Ex. 33, at 140:18-141:8, 188:23-189:16 (Gardener  
 8 MDL Tr.). But this conclusion is contrary to the concessions Plaintiffs' experts make regarding the  
 9 differences in physiology between the prenatal and postnatal periods. *See* Defs.' Br. No. 3, at 32-  
 10 33. For example, Plaintiffs' experts concede that brain development differs during each month of  
 11 infant development and as a result, heavy metal exposure will have different effects depending on  
 12 the timing of exposure. *See id.*; *see, e.g.*, Kiser Decl., Ex. 26, at 48:3-10 (Ritz MDL Tr.) ("Q. Okay.  
 13 And if I'm understanding you correctly, is it fair to say that lead exposure can have different effects  
 14 depending on the timing of that exposure? A. Correct. Q. Okay. And is the same true for inorganic  
 15 arsenic? A. Yes.").

16       Additionally, Plaintiffs' experts agree that the methodologically appropriate period to  
 17 examine for purposes of general causation is the postnatal period when baby food is consumed. *See*  
 18 Kiser Decl., Ex. 26, at 43:19-25 (Ritz MDL Tr.) (stating that the relevant period for the causation  
 19 question in this litigation is "during neurodevelopment," i.e., "after the baby is born and before the  
 20 diagnosis is made."); *see also* Kiser Decl., Ex. 35, at 83:25-84:4 (Guilarte MDL Tr.) ("**Q. And in  
 21 order to determine if a substance can cause ASD postnatally, one should look at studies of  
 22 postnatal exposure, correct? A. Correct. Q. Okay. And the same is true for ADHD obviously,  
 23 correct? A. Correct.**") (emphasis added).

24       When an expert relies on studies conducted under conditions materially different from those  
 25 at issue in the litigation—as Plaintiffs' experts do here—the proffering party must demonstrate that  
 26 the method of extrapolation is generally accepted. *Schudel v. GE*, 120 F.3d 991, 997 (9th Cir. 1997);  
 27 *Bextra*, 524 F. Supp. 2d at 1180. Improper extrapolation, "[t]hat is, leaping from an accepted  
 28 scientific premise to an unsupported conclusion," is a classic Rule 702 "red flag." *Downs v. Perstorp*

1 *Components, Inc.*, 126 F. Supp. 2d 1090, 1125 (E.D. Tenn. 1999).<sup>19</sup> Here, the relevant period is the  
 2 postnatal period when children consume baby food. Rule 702 therefore prohibits Plaintiffs' experts'  
 3 speculative assumption that studies on the alleged effects of lead and arsenic exposure during the  
 4 prenatal period can reliably support their opinions.

5 **3. Plaintiffs Fail to Justify Extrapolation from Non-U.S. Population Data.**

6 Plaintiffs' experts also fail to justify their extrapolation from numerous studies and meta-  
 7 analyses that evaluate heavy metal exposures and autism or ADHD in populations outside the United  
 8 States to draw conclusions regarding exposures in the U.S. population. *See Opp.* at 88-91. Plaintiffs  
 9 do not address the fact that the vast majority of the studies included in the meta-analyses do not  
 10 satisfy temporality or assess diagnosed autism or ADHD. *See* *Defs.*' Br. No. 3, at 38. Nor do  
 11 Plaintiffs explain their own expert's admission that children's exposures in non-western and/or non-  
 12 industrialized nations that are the subject of the studies their experts rely upon "are much higher  
 13 than children's exposures in the U.S." and that "the heavy metal studies that are most relevant to the  
 14 US population . . . are studies from the United States." Kiser Decl., Ex. 35, at 121:6-122:1, 128:23-  
 15 129:2 (Guilarte MDL Tr.).

16 Plaintiffs' response to Defendants' argument is twofold. First, they claim that "lead is a  
 17 neurotoxin no matter where you are" and that "[t]he ability of lead and arsenic to penetrate the  
 18 [blood-brain barrier] of an infant and interfere with neurodevelopment is not constrained by  
 19 geography." *Opp.* at 88. But the problem is not one of geography *per se*—rather, it is the  
 20 dramatically higher levels of lead and arsenic exposures in these non-U.S. populations, which are  
 21 not reflective of lead or arsenic levels in U.S. children consuming Defendants' baby food products.

22 Second, Plaintiffs attempt to blur the gap between U.S. and non-U.S. populations by arguing  
 23

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24 <sup>19</sup> Plaintiffs offer a single case, *Junk v. Terminix Int'l Co.*, 577 F. Supp. 2d 1086 (S.D. Iowa 2008),  
 25 for the general proposition that "there is nothing improper, scientifically, with considering prenatal  
 26 data." *Opp.* at 92. Plaintiffs note that the expert in *Junk* provided testimony regarding a "chemical's  
 27 ability to cause neurodevelopmental harm based on prenatal and postnatal studies" (*id.*), but the case  
 28 is plainly distinguishable. In *Junk*, **both** in utero and postnatal exposure to the relevant chemical  
 (chlorpyrifos) were at issue at the general causation stage. *See id.* at 1092 ("[T]he proper inquiry in  
 the general causation analysis is whether in utero and postnatal exposure to chlorpyrifos is capable  
 of causing severe developmental delay.").

1 that there are meta-analyses that include both U.S. and non-U.S. populations.<sup>20</sup> Defendants have  
 2 already explained how many of the studies in these meta-analyses (e.g., Stojasavljevic (2023))  
 3 included non-US populations where the lead or arsenic exposure levels were dramatically higher  
 4 than those in U.S. children—in some cases by a factor of six.<sup>21</sup> And Plaintiffs’ argument in favor of  
 5 meta-analyses such as Ding (2023), Shiani (2023), and Nakhaee (2022)—on the grounds that they  
 6 examined both U.S. and non-U.S. data—is undermined by those meta-analyses’ findings of **no**  
 7 *statistically significant increase in lead levels in children with autism in the U.S. or North*  
 8 *American populations.* See Defs.’ Br. No. 3, at 36-38; Kiser Decl., Ex. 64 (Ding (2023)) (evaluating  
 9 the results of studies in North America, as opposed to different populations, and concluding that  
 10 lead levels were *not* statistically significantly higher in children with autism *in North America* as  
 11 compared to controls); Kiser Decl., Ex. 65, at 8-9 (Table 3), 10 (Figure 3) (Shiani (2023)) (reporting  
 12 either decreased lead levels or no statistically significant difference in lead levels in autism cases in  
 13 each of the six studies conducted in the U.S.); Kiser Decl., Ex. 43, at 311:20-313:24 (Ritz Landon  
 14 Tr.) (stating that Nakhaee (2022) reports that of the three studies conducted in North America, two  
 15 found no association between blood lead level and autism, and one found *lower* hair lead levels in  
 16 children with autism). Faced with data from U.S. populations that undermines their argument,  
 17 Plaintiffs’ experts cannot reliably extrapolate from non-U.S. populations.

18           **D. Plaintiffs’ Experts Rely on Studies That Do Not Satisfy Temporality and/or Do**  
 19           **Not Establish Causation.**

20           As explained in Defendants’ Motion, Plaintiffs’ experts overwhelmingly rely on studies that  
 21 do not satisfy the requirement of temporality. Defs.’ Br. No. 3, at 40-42. In fact, of the 114 lead and  
 22

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23           <sup>20</sup> Plaintiffs also argue that non-U.S. studies are useful because “[i]n observational epidemiology,  
 24 the focus is on relative risk . . . in the context of lead and arsenic, how the greater exposed group  
 25 compares to the lesser exposed group” and therefore “if the overall baseline exposure is higher in  
 26 one country, it will be higher in both groups—the study, thus, reflects whether differences of metal  
 27 exposure within comparable groups increases the risk of a disease outcome.” Opp. at 91. But if that  
 28 were true, we would then expect to see directionally similar relative risks for U.S. and non-U.S.  
 populations, but the data show the opposite.

27           <sup>21</sup> Notably, Plaintiffs fail to respond to, and thereby concede, Defendants’ argument that all of the  
 28 meta-analyses on which Plaintiffs’ experts rely have very high statistical heterogeneity, which renders  
 them unable to reliably support a causation opinion. See Defs.’ Br. No. 3, at 38 n.16.

1 61 arsenic studies cited by Plaintiffs' experts, *only 13 and 7 studies*, respectively, met the  
 2 temporality criterion. In other words, *nearly 90% of these studies cannot satisfy temporality*,  
 3 underscoring the serious analytical gap between Plaintiffs' experts' opinions and the data on which  
 4 those experts rely. And the very few studies on which Plaintiffs' experts rely that do satisfy  
 5 temporality and examine at diagnosed autism/ADHD can never reliably establish causation. Thus,  
 6 even ignoring Plaintiffs' extrapolation problems, Plaintiffs' experts' opinions should be excluded.

7 **1. Plaintiffs' Experts Cannot Justify Their Improper Reliance on Studies  
 8 That Do Not Establish Temporality.**

9 Plaintiffs concede—as they must—that an epidemiological study must satisfy temporality to  
 10 support a causation opinion. Indeed, the case law, the Reference Manual on Scientific Evidence, and  
 11 Plaintiffs' own experts admit that this essential requirement must be present to make causal inferences.  
 12 Defs.' Br. No. 3, at 40-41. Yet after laying down this marker as part of their methodology, Plaintiffs'  
 13 causation experts cast it aside. They readily admit to basing their opinions almost entirely on cross-  
 14 sectional studies or case-control studies that do not and cannot satisfy temporality.<sup>22</sup> *See* Defs.' Br.  
 15 No. 3, at 40-42; *see also* Kiser Decl., Ex. 7, at 60 (Gardener Rep.) (stating that the studies measured  
 16 arsenic in children “after the diagnosis had already occurred.”). For example, Plaintiffs' Opposition  
 17 shows a forest plot from Dr. Gardener's report in which 11 of the 16 cited studies concerning lead  
 18 exposure<sup>23</sup> failed to establish temporality, a fact which Dr. Gardener concedes. *Compare* Opp. at 16  
 19 *with* Kiser Decl., Ex. 7, at 69 (Gardener Rep.) (discussing how “most of these studies” in her forest  
 20 plot measured blood lead levels “after ADHD had been diagnosed”). This same problem infects the  
 21 systematic reviews and meta-analyses Plaintiffs cite, such as Wang (2019), Ding (2023), and Dalla  
 22

23 Plaintiffs' Opposition mistakenly argues that Defendants' brief only focused on cross-sectional  
 24 studies. Opp. at 93-97. Not so. Defendants also focused on case-control studies that failed to  
 25 establish temporality. Defs.' Br. No. 3 at 39-42.

26  
 27 <sup>22</sup> These studies include Ha 2008 (Pltfs. Ex. 85); Joo 2017 (Kim Decl., Ex. 5); Kim 2010 (Kim Decl.,  
 28 Ex. 6); Zhang 2015 (Kim Decl., Ex. 7); Braun 2006 (Kim Decl., Ex. 8); Froehlich 2009 (Kim Decl.,  
 Ex. 9); Kim 2013 (Kim Decl., Ex. 10); Munoz 2020 (Kim Decl., Ex. 11); Park 2016 (Kim Decl.,  
 Ex. 12); Wang 2008 (Kim Decl., Ex. 13); and Yousef 2011 (Kim Decl., Ex. 14).

1 (2022). *See* Opp. at 18-19, 61-62, 90-91.<sup>24</sup> Plaintiffs fail to cite a single case holding that  
 2 epidemiological studies that do not establish temporality can reliably be used to support a general  
 3 causation opinion.

4 Plaintiffs also fail to offer any scientific justification for the fact that the failure to satisfy  
 5 temporality infects almost 90% of the studies their experts cite. Recognizing that the cross-sectional  
 6 and case-control studies on which their experts rely fail to show that the lead or arsenic exposure  
 7 occurred before the outcome assessed, Plaintiffs offer two arguments for why these studies  
 8 nonetheless “provide valuable evidence.” Opp. at 93. But these arguments only underscore their  
 9 experts’ willingness to offer result-oriented conclusions.

10 **First**, Plaintiffs argue that lead may be stored in the bones and released later into the blood,  
 11 such that studies measuring BLLs could possibly capture “exposures *predating* disease onset.” Opp.  
 12 at 94. This is facially speculative. Not a single study Plaintiffs cite claims that lead measured in the  
 13 blood of a child or adolescent is capturing exposures from years earlier—let alone from the specific  
 14 window when baby food is eaten. And it is even more speculative to suggest that biomarker lead  
 15 levels capture exposures *predating* the onset of autism or ADHD. Moreover, Plaintiffs’ own  
 16 toxicology expert, Dr. Aschner, disagrees with this claim. He testified that a BLL captures a window  
 17 of exposure of one or two months and the fact that lead can be stored in bone is “not relevant for  
 18 kids because the kids themselves probably do not release the lead from bone.” Pltfs. Ex. 105, at  
 19 130:12-16 (Aschner N.C. Tr.). Thus, no lead would be expected to leach from bones so as to then  
 20 be measured in the blood in any of the studies at issue. *Id.* at 134:19-135:4. Plaintiffs’ own experts  
 21 concede that biomarker testing reflects only recent levels. According to Dr. Aschner, BLLs  
 22 “basically tell you if someone has been exposed in recent times, one month, two months, yes.” *Id.*

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23  
 24 <sup>24</sup> Kiser Decl., Ex. 64, at 12 (Ding (2023)) (“the included studies are case-control design so that a  
 25 specific causality could not be determined between heavy metals exposure and ASD occurrence.”);  
 26 Pltfs. Ex. 39, at 1907-12 (Wang (2019)) (Tables 1 and 2 listing studies included in meta-analysis  
 27 which include case-control and cross-sectional studies that do not establish temporality); *id.* at 1917  
 28 (“Future studies are recommended to investigate prenatal exposure to heavy metals in a cohort study  
 design for determining causal relationships.”); Pltfs. Ex. 93, at 4-10 (Dalla (2022)) (Table 1  
 including the cross-sectional studies cited by Plaintiffs, Arbuckle 2016, which did not establish  
 temporality); Kim Decl., Ex. 15, at 240-42 (Guo (2019)) (Table 1 listing studies included in meta-  
 analysis which include case-control and cross-sectional studies that do not establish temporality).

1 at 139:16-21; *see also* Kiser Decl., Ex. 7, at 48 (Gardener Rep.) (“[l]ead measured in blood and  
 2 urine reflects **short-term, recent exposure.**”) (emphasis added); Kiser Decl., Ex. 4, at 4 (Hu Rep.)  
 3 (stating BLLs provide a ““snapshot”” of lead exposure representative of “several weeks.”). Thus,  
 4 BLLs measured in the studies cannot speak to anything but perhaps two months of exposure, nothing  
 5 more. They cannot reliably “capture pre-disease” data, as Plaintiffs claim. Opp. at 94.<sup>25</sup>

6 **Second**, Plaintiffs argue that the existence of a small number of studies that *do* meet the  
 7 temporality criterion “ameliorates” the methodological flaw in drawing causal conclusions from the  
 8 vast majority that do not. Opp. at 94-95. Again, outside of their own experts’ say-so, Plaintiffs cite  
 9 no scientific authority to support this claim. *See id.* And even Plaintiffs’ experts concede that  
 10 temporality is a “component that is necessary for making a causal contribution.” Defs.’ Br. No. 3,  
 11 at 40 (quoting Dr. Ritz’s Rep.); *see also* Kiser Decl., Ex. 35, at 63:15-20 (Guilarte MDL Tr.)  
 12 (admitting that temporality is “a required component of determining causality”). Pointing to a small  
 13 number of studies, such as Kim 2016 or Arora 2017, that do establish temporality (*see* Opp. at 95)  
 14 cannot transform the studies that do not into “valuable evidence.” *See* Opp. at 93.

15 Plaintiffs’ experts’ reliance on studies that cannot establish temporality is particularly  
 16 methodologically unsound here given the significant concerns regarding reverse causality. *See*  
 17 Defs.’ Brief at 40-42. Plaintiffs dismiss this argument as speculative, which is curious given that  
 18 some of their *own experts* agree with Defendants. Dr. Guilarte testified that “[p]ica and prolonged  
 19 hand-to-mouth behaviors may be one reason why kids with ASD have elevated levels of heavy  
 20 metals” and that “[i]n epidemiological studies on heavy metals and ASD, it is important to control  
 21 for pica as a potential confounder to help control for reverse causality.” Kiser Decl., Ex. 35, at  
 22 157:14-17, 158:2-9 (Guilarte MDL Tr.); *see also* Kiser Decl. Ex. 34, at 103:14-21 (Aschner MDL  
 23 Tr.) (acknowledging that children with autism are at increased risk of pica).<sup>26</sup>

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24  
 25 <sup>25</sup> The same holds true for urine as a biomarker. It can only about a month of exposure (if that). Kim  
 26 Decl., Ex. 2, at 83:3-10 (Feb. 4, 2022, N.C. Hrg. Tr.). For hair, one centimeter of hair measured  
 from the scalp is about one month’s worth of exposure. *Id.* at 83:22-26.

27 <sup>26</sup> Plaintiffs’ reliance on the Brown (2025) and Adams (2006) studies is inapposite. *See* Opp. at 96.  
 28 Brown 2025 was limited by its sample size (a total of only 14 children with pica), and the authors  
 acknowledged that “[t]here was no standardized question for pica in the history taking, or obligation

1 Dr. Guilarte also agreed that “low levels or deficiencies in essential nutrients—may be the  
 2 reason why kids with ASD have elevated levels of lead and arsenic in their biomarkers” and that it  
 3 is “important to control for levels of essential nutrients like iron, zinc and calcium in epidemiological  
 4 studies of heavy metals and ASD.” *Id.* at 202:14-22; 208:10-20. Thus, Plaintiffs’ own expert agreed  
 5 that there are significant concerns about reverse causality in studies of heavy metals and ASD, which  
 6 makes establishing temporality in this body of literature all the more critical.

7 **2. The Few Studies on Which Plaintiffs’ Experts Rely That Satisfy  
 8 Temporality, Consider Postnatal Exposure, and Examine Diagnosed  
 Autism and ADHD Do Not Reliably Establish Causation.**

9 As noted above, Plaintiffs’ experts cite very few studies that meet temporality, examine a  
 10 postnatal exposure window, and use an endpoint of diagnosed autism and ADHD. For arsenic and  
 11 diagnosed autism there are no studies at all that meet temporality and examine postnatal exposure.  
 12 For lead and diagnosed autism there are only three. For lead and diagnosed ADHD there is only  
 13 one. And Plaintiffs’ excuses and sleights of hand cannot make this limited and flawed data set  
 14 anything more than it is—not enough for experts to reliably extrapolate to a claim that consuming  
 15 baby food causes autism and ADHD.

16 **a) Studies of Arsenic and Autism.**

17 Because they have no studies evaluating postnatal exposure to arsenic and autism that satisfy  
 18 temporality, Plaintiffs argue that three *prenatal* studies (Skogheim, Dou, and Long) provide the  
 19 foundation for their experts’ opinions that exposure to arsenic can cause autism. Even if prenatal  
 20 studies were relevant to this case—which they are not—Plaintiffs’ discussion of the results of these  
 21 studies is misleading. For example, Plaintiffs misleadingly quote from the Long study but fail to cite  
 22 the study’s most important finding: the authors found ***no statistically significant association***

23  
 24 to record the presence or absence of the behaviour in the clinical notes.” Plts. Ex. 106, at 5 (Brown  
 25 2025). Additionally, the study was conducted in the UK where environmental heavy metal exposure  
 26 (e.g., via lead paint etc., that a child with pica may ingest) is exponentially smaller than in the  
 27 developing countries, where the vast majority of studies on which Plaintiffs’ experts rely were  
 28 conducted. *See id.* at 1-2. Similarly, the Adams (2006) study included a total of only 16 children  
 with pica. The authors of that study, conducted in the United States, concluded that “[o]verall, it  
 appears that the children with autism ***do not have major differences in their levels of toxic metals***  
 compared to controls.” Plts. Ex. 107, at 204 (Adams 2006) (emphasis added).

1 between arsenic exposure and autism. Kiser Decl., Ex. 66, at 10 (Long (2019)). "Similar levels of  
2 elements and heavy metals between ASD cases and controls were observed."<sup>27</sup> *Id.*

Even worse, Plaintiffs outright misrepresent Dou (2024); the Dou authors conclude that “[a] doubling in arsenic was associated with ***lower ASD risk*** (RR = 0.84, 95% CI 0.74, 0.94)” and was still statistically significantly decreased even after adjusting for multiple comparisons. Kiser Decl., Ex. 71, at 6 (Dou (2024)) (emphasis added). Using ellipses, Plaintiffs also cite Dou to misleadingly suggest that the authors found an association with prenatal arsenic exposure when their finding related to cadmium and cesium. Opp. at 98. Finally, in Skogheim, the statistically significant increased risk for arsenic was limited to the 2<sup>nd</sup> quartile of exposure and there was no statistically significant increased risk for the higher (3<sup>rd</sup> and 4<sup>th</sup>) quartiles of exposure. Indeed, the highest arsenic exposure level in Skogheim was in the direction of a non-statistically significantly *decreased* risk. These findings are inconsistent with causation. *See* Federal Judicial Center, Reference Manual on Scientific Evidence, at 603 (3d ed. 2011), [Reference Manual on Scientific Evidence, Third Edition \(2011\)](#) (“[a] dose–response relationship means that the greater the exposure, the greater the risk of disease. Generally, higher exposures should increase the incidence (or severity) of disease.”).

**b) Studies of Lead and Autism.**

17 Plaintiffs do not dispute that there are only three studies evaluating postnatal exposure to  
18 lead and diagnosed autism that satisfy temporality (Frye, Arora, and Abdullah). *See Opp.* at 98-99.  
19 Nor do Plaintiffs dispute that the Frye and Abdullah studies showed ***no association between lead***  
20 ***exposure and autism.*** *See id.* Instead, Plaintiffs suggest that the Arora study shows an association  
21 between postnatal lead exposure and autism. *Id.* In fact, Plaintiffs never respond to Defendants'  
22 argument and their own expert's admission that "***[a]fter correction for multiple comparisons, there***  
23 ***were no statistically significant associations at any point in time between lead levels and ASD in***

<sup>26</sup> Plaintiffs misleadingly quote from this article by using ellipses to remove key words. The  
complete quote reads: “The present study showed that environmental endocrine disrupting  
27 chemicals (EDCs), such as PFAS, some metals, and their biological activities can be detected in  
amniotic fluid, indicating that EDCs can cross the placenta and increase the potential fetal exposure  
28 to these environment contaminants. EDCs might modify ASD risk by influencing the hormone  
receptor function.” Kiser Decl., Ex. 66, at 16 (Long (2019)).

1 ***the Arora study.***” Kiser Decl., Ex. 35, at 237:20-238:6 (Guilarte MDL Tr.) (emphasis added); *see*  
 2 *also id.* at 74:21-75:1 (acknowledging that “when multiple comparisons are performed in an  
 3 epidemiological study, for example, a Bonferroni correction should be used to decrease the  
 4 likelihood of finding false associations due to chance”). Dr. Ritz’s rank speculation that the Arora  
 5 authors “probably didn’t have enough statistical power to show the other effects, if there were some”  
 6 cannot supplant the lack of an observed association between postnatal lead exposure and autism in  
 7 the Arora study itself. Pltfs. Ex. 24, at 213:7-214:11 (Ritz N.C. Tr. Vol. I). That is, an expert cannot  
 8 ***presume*** a study would demonstrate an effect, if sufficiently powered, where the actual results  
 9 showed no association.

10 With respect to the four studies evaluating *prenatal* exposure to lead and diagnosed autism  
 11 that satisfy temporality, Plaintiffs concede that three of the studies (Long, Skogheim, and Wegmann)  
 12 show no statistically significant association. Opp. at 99. Still, Plaintiffs claim that “it is improper to  
 13 discount studies observing an association between exposure and outcome merely because of the lack  
 14 of statistical significance.” Opp. at 99. But Plaintiffs’ own expert conceded that lack of statistical  
 15 significance means there is no reliable association. Kiser Decl., Ex. 35, at 73:16-74:2 (Guilarte MDL  
 16 Tr.) (“Q. But based on my review of the studies—of your studies, your published work, when the  
 17 results in your studies are not statistically significant, you conclude that there’s no difference  
 18 between groups or no association, correct? THE WITNESS: Correct. Q. And that’s just a generally  
 19 accepted and reliable statistical methodology, correct? THE WITNESS: Correct.”). If Plaintiffs’  
 20 experts seek to rely on non-statistically significant findings to support a causation opinion, they must  
 21 offer reliable scientific analysis as to why that is appropriate and also place as much weight on non-  
 22 statistically significant findings that show a *decreased* risk. They have done neither.

23 As for the Dou study, Plaintiffs misstate Defendants’ argument. Dou is a single, unreplicated,  
 24 study with inconsistent results (finding no association with maternal urinary lead levels, but a small  
 25 association with maternal blood lead levels). *See* Defs.’ Br. No. 3, at 44-45. Plaintiffs’ Opposition  
 26 does not address *any* of these facts. Plaintiffs respond only to the fact that Dou *also* did not adjust for  
 27 multiple comparisons by stating that “Dr. Guilarte never faulted Dou (2024) for not adjusting.” Opp.  
 28 at 99. But the fact that Dr. Guilarte did not fault the Dou study specifically is irrelevant given his

1 unequivocal testimony that “when multiple comparisons are performed in an epidemiological study,  
 2 for example, a Bonferroni correction **should** be used to decrease the likelihood of finding false  
 3 associations due to chance.” Kiser Decl., Ex. 35, at 74:21-75:1 (Guilarte MDL Tr.) (emphasis added).

4 **c) Studies of Lead and ADHD.**

5 As for lead exposure and ADHD, Plaintiffs fail to identify any study other than Ji (2018)  
 6 that is postnatal and satisfies temporality, and Ji shows *no association* with blood levels of less than  
 7 5 ug/dL. Opp. at 99; *see* Kiser Decl., Ex. 91, at 2 (Ji (2018)). And as much as Plaintiffs try to dispute  
 8 this fact, they cannot escape their own expert’s acknowledgement of this fact. Kiser Decl., Ex. 35,  
 9 at 295:7-11 (Guilarte MDL Tr.) (“Q. And my question -- my first question is there was no  
 10 association in this study for blood lead levels of less than 5 micrograms per deciliter, correct? THE  
 11 WITNESS: That’s what this table says.”). Plaintiffs also do not dispute their expert’s testimony that  
 12 “it would be important” to control for socioeconomic status (“SES”) in epidemiological studies of  
 13 heavy metals and neurodevelopmental outcomes and that Ji did not control for SES. Instead,  
 14 Plaintiffs contend that some other, *unnamed* studies did control for SES and purportedly found an  
 15 association. Opp. at 100. Even if that were true (and Plaintiffs do not identify any such studies), that  
 16 would have no bearing on the limitations of the Ji study, which is the only study that satisfies  
 17 temporality and assesses postnatal lead exposure and diagnosed ADHD.

18 Finally, Plaintiffs again argue that their experts are entitled to offer causal opinions based on  
 19 reported associations that are not statistically significant. Opp. at 100. But again, Plaintiffs’ own  
 20 experts agree that statistical significance is the generally accepted methodology for determining  
 21 whether a study’s findings are due to chance versus a reliable, replicable association. Kiser Decl.,  
 22 Ex. 35, at 72:6-10 (Guilarte MDL Tr.); Kiser Decl., Ex. 43, at 117:4-14, 136:8-12 (Ritz Landon R.  
 23 Tr. Vol. I); Kiser Decl., Ex. 29, at 90:15-92:9 (Hu MDL Tr.). As Plaintiffs’ expert Dr. Guilarte  
 24 testified, results that are not statistically significant are interpreted as showing “no association,” and  
 25 this is “just a generally accepted and reliable statistical methodology.” Kiser Decl., Ex. 35, at 72:15-  
 26 19, 73:16-74:2 (Guilarte MDL Tr.); *see also In re Zoloft (Sertraline Hydrochloride) Prods. Liab.*  
 27 *Litig.*, 26 F. Supp. 3d 449, 456 (E.D. Pa. 2014) (courts should not be “pioneers, forging new trails  
 28 in scientific thinking, especially when that means departing from well-established research

1 principles, such as the principle of statistical significance"). Plaintiffs' citation to *Hardeman v.*  
 2 *Monsanto Company*, 997 F.3d 941 (9th Cir. 2021), does not assist them. *See* Opp. at 100. Far from  
 3 suggesting that an expert can rely entirely or largely on studies that do not show a statistically  
 4 significant association, the court concluded only that the loss of statistical significance in sensitivity  
 5 analyses adjusting for a potential confounder did not alone bar the expert's opinion. *Id.* Here, in  
 6 contrast, Plaintiffs do not have studies that actually adjust for critical confounding variables, such  
 7 as genetics. And Plaintiffs' experts relied on non-statistically significant findings of increased risk,  
 8 while ignoring non-statistically significant (or even statistically significant) *decreased* risks. Defs.'  
 9 Br. No. 3, at 46-47. Plaintiffs cannot justify that form of unreliable cherry-picking, especially when  
 10 their own experts recognize the importance of statistical significance.

11 In short, Plaintiffs argue that their experts (1) can use the studies without temporality to  
 12 support the ones with temporality; (2) can use the studies without diagnoses to support the ones with  
 13 diagnoses; and (3) use the studies without the relevant postnatal exposure to support the ones with  
 14 postnatal exposures. But it is undisputed that there are no studies that evaluate postnatal exposure  
 15 to arsenic and diagnosed autism that satisfy temporality. And while Plaintiffs champion Arora  
 16 (2017) as the one study that satisfies temporality and purportedly shows a statistically significant  
 17 increased risk between postnatal exposure to lead and diagnosed autism, Plaintiffs' own expert  
 18 agreed that Arora does not show a statistically significant increased risk. Nor do Plaintiffs identify  
 19 any postnatal study of lead and diagnosed ADHD that meets temporality, controls for confounders  
 20 that their own expert said must be controlled for and shows an association at levels of exposure  
 21 relevant to this litigation.

22 **III. CONCLUSION**

23 For the foregoing reasons, Defendants respectfully request that the Court exclude the general  
 24 causation opinions of Drs. Aschner, Gardener, Guilarte, Hu, Ritz, and Shapiro.

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**CERTIFICATE OF SERVICE**

I certify that on November 7, 2025, I electronically filed the foregoing **DEFENDANTS' REPLY IN SUPPORT OF DEFENDANTS' MOTION TO EXCLUDE PLAINTIFFS' CAUSATION/ EPIDEMIOLOGY EXPERTS** using the ECF system, which sent notification of such filing to all counsel of record.

/s/ Brooke K. Kim

Brooke K. Kim